

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant(s) : Toshiyuki Takai, et al.  
U.S. Appln. No. : 10/712,118  
U.S. Filing Date : November 13, 2003  
Title of Invention : NON-HUMAN ANIMAL MODEL OF OLIGODENDROCYTE  
DEVELOPMENTAL DISORDER  
Confirm No. : 8301  
Examiner : Joanne Hama, Ph.D  
Art Unit : 1632

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**PRELIMINARY AMENDMENT ACCOMPANYING RCE**

**Mail Stop RCE**

Commissioner for Patents  
P.O. Box 1450  
Alexandria, VA 22313-1450

Dear Sir:

This Preliminary Amendment is being filed with a Request for Continued Examination (RCE). As the RCE contains a Request for Extension of Time, it is believed that no fees are due by entry of this paper. However, the Commissioner is hereby authorized to charge any additionally required fee, or credit any overpayment in fees, to Deposit Account No. 50-0320.

**AMENDMENT IN THE CLAIMS:**

Kindly amend the claims, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, to read as follows:

1. (Currently amended) A transgenic mouse model showing ~~hypomyelinosi~~s of the thalamus that can be a cause of Nasu-Hakola disease, and showing a neuropsychiatric disorder caused by the hypomyelinosi, wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function and shows hypomyelinosi of the thalamus, and wherein the homozygous disruption includes the promoter region and exons 1, 2, and 3.

2. (Canceled)

3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheontotypically exhibited as a myelinogenesis developmental disorder ~~that can be a cause of Nasu-Hakola disease~~.

4.(Currently amended) The transgenic mouse model of claim 1 or 3, ~~wherein the neuropsychiatric disorder caused by hypomyelinosi is further showing a disease~~ selected from the group consisting of ~~Nasu-Hakola disease caused by hypomyelinosi~~, dementia caused by ~~hypomyelinosi~~ Nasu-Hakola disease, schizophrenia caused by ~~hypomyelinosi~~ Nasu-Hakola disease, schizotypal personality disorders caused by ~~hypomyelinosi~~ Nasu-Hakola disease, obsessive-compulsive disorders caused by ~~hypomyelinosi~~ Nasu-Hakola disease, or Tourette's syndrome caused by ~~hypomyelinosi~~ Nasu-Hakola disease.

5. (Currently amended) The transgenic mouse model of claim 1 or 3, ~~wherein the neuropsychiatric disorder caused by hypomyelinosi is Nasu-Hakola disease caused by hypomyelinosi or further showing~~ dementia caused by ~~hypomyelinosi~~ Nasu-Hakola disease.

6-18. (Canceled)

19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.

20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.

**REMARKS**

Reconsideration and withdrawal of the rejections of this application and consideration and entry of this paper are respectfully requested in view of the herein remarks and accompanying information, which place the application in condition for allowance.

**I. STATUS OF CLAIMS AND FORMAL MATTERS**

Claims 1, 3-5, 19, and 20 are currently under consideration. Claims 1 and 3-5 are amended without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents.

No new matter is added.

It is submitted that the claims herewith are patentably distinct over the prior art, and these claims are in full compliance with the requirements of 35 U.S.C. §112. The amendments to the claims presented herein are not made for purposes of patentability within the meaning of 35 U.S.C. §§§§ 101, 102, 103 or 112. Rather, these amendments and additions are made simply to clarify the scope of protection to which Applicant is entitled.

Support for the amended claims can be found throughout the specification as originally filed. For instance, support for amended claims 1 and 3-5 can be found, as an example, in the paragraphs beginning on page 4, line 2, on page 6, line 3, and on page 7, line 24, and in the original claims.

**II. REJECTIONS UNDER 35 U.S.C. § 112 ARE OVERCOME**

Claims 1, 3-5, 19, and 20 are maintained as rejected under 35 U.S.C. § 112, first paragraph, as failing to comply with the enablement requirement. The rejection is respectfully traversed.

According to the Office Action, Applicants' previous amendments and arguments were not persuasive; the amended claims could allegedly be read such that the hypomyelinosi could not only be a cause of Nasu-Hakola disease, but various other neurological diseases as well. The Office Action contends that there is nothing in the art or specification providing guidance that hypomyelinosi was a model of other diseases which results in the disorders disclosed in claim 4.

Applicants respectfully draw attention to the claim amendments herein. Importantly, claim 1 is clarified as relating to a transgenic mouse model “showing Nasu-Hakola disease, wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function and shows hypomyelinos of the thalamus, and wherein the homozygous disruption includes the promoter region and exons 1, 2, and 3.” Further, the subject matter relating to neuropsychiatric disorders in claims 4 and 5 is clarified as “being caused by Nasu-Hakola disease.”

In view of the claim amendments, Applicants argue that the instant claims are enabled by the specification. The scope of the claims are clarified as a result of the claim amendments, such that the claims are herein relate to a transgenic mouse model showing Nasu-Hakola disease and that shows neuropsychiatric disorders caused by Nasu-Hakola disease. There is also ample guidance and working examples in the specification for a transgenic mouse showing Nasu-Hakola disease, notably in Examples 1 and 3-5, such that undue experimentation would not be necessary to make/use the present invention. The prior art, as recited in the specification, indicates that Nasu-Hakola disease is associated with loss of function of DAP 12 (page 2, lines 18-29 of the specification). Furthermore, in the Final Office Action mailed April 23, 2007, the Examiner admitted that the specification is enabling for “a transgenic mouse comprising a homozygous disruption of DAP 12 (DNAX Activation Protein 12) in its genome, wherein the transgenic mouse exhibits hypomyelinos of the thalamus, and wherein the mouse exhibits Nasu-Hakola disease” (page 2, sixth paragraph). Regarding claims 4 and 5, the Examiner suggested that “[r]ather than have the neuropsychiatric disorders be associated with disruption in DAP12 gene function, the neuropsychiatric disorders could be associated with Nasu-Hakola disease” (page 5, first paragraph).

Therefore, in consideration of instant claims, Applicants’ remarks, and the indications made by the Office Action, it is asserted that the instant claims are enabled by the specification. Accordingly, reconsideration and withdrawal of the rejection under § 112, first paragraph, is respectfully requested.

**REQUEST FOR INTERVIEW**

If any issue remains as an impediment to allowance, an interview with the Examiner and SPE are respectfully requested and the Examiner is additionally requested to contact the undersigned to arrange a mutually convenient time and manner for such an interview.


**CONCLUSION**

In view of the remarks and amendments herewith, the application is in condition for allowance. Favorable reconsideration of the application and prompt issuance of a Notice of Allowance are earnestly solicited. The undersigned looks forward to hearing favorably from the Examiner at an early date, and, the Examiner is invited to telephonically contact the undersigned to advance prosecution.

Respectfully submitted,

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